Diagnosis and Endovascular Treatment of Vertebral Arteriovenous Fistulas in Neurofibromatosis Type 1

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Summary

We present diagnostic problems, strategies, techniques and material selection for endovascular treatment of high flow arteriovenous fistula (AVF) of tortuous and fragile vertebral artery (VA) with neurofibromatosis type 1 (NF1).

Diagnosis of NF1 was easy in four of our cases because of neurofibromatosis, skin pigmentation and various skeletal abnormalities. These stigmas of NF1 were lacking in one case, and the only clue to the diagnosis was ovoid bone defects of the skull vault. Diagnosis was made by performing biopsy of scalp neurofibromas incidentally found on CT. In two initial cases, venous varix were packed with coils by transvenous approach after the transarterial embolisation failed to completely cure the fistula. In three recent cases, blood flow through the fistula was markedly reduced as an initial step by placing detachable coils into the distal and proximal stumps of the afferent VA. Then a liquid adhesive was injected under systemic hypotension to completely occlude the fistula.

Control angiography revealed that the AVFs were completely occluded in all cases. Long-term angiographical and clinical status have been stable in all cases.

Trying to attain complete occlusion of fistulas using detachable balloons is not an appropriate treatment option for high flow fistulas situated on markedly dilated, tortuous and fragile VAs of patients with NF1. Also, trapping of fistulas is not justified because of the numerous potential feeding pedicles, and makes the following procedure difficult.

Introduction

Vertebral AVFs are abnormal communications between the extracranial VA or its branches and the surrounding venous plexus [1]. Symptoms often related to vertebral AVFs are bruit and neck pain, but brain and spinal cord dysfunction due to steal phenomenon, venous hypertension, or medullary compression by dilated veins have also been reported [2,3]. Recently, intraventricular haemorrhage due to high-flow vertebral AVF was reported (F. Simionato, poster presentation at the World Federation of Interventional and Therapeutic Neuroradiology, Vilamoura, Portugal, 1999). These rare lesions can be either traumatic or spontaneous in origin. Traumatic lesions with a known cause may be the result of penetrating or blunt trauma or of iatrogenic origin (commonly direct intentional puncture of a VA for diagnostic angiography and unintentional puncture during

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insertion of a jugular catheter). Spontaneous lesions have been associated with Marfan's syndrome, progeria, fibromuscular dysplasia, and hereditary connective tissue disorders such as Ehlers-Danlos syndrome type 4 and NF1 [4-7].

Neurofibromatosis is a congenital and familial disease, currently divided into two distinct types with autosomal disorder [6,8]. The NF1 has an abnormality in the long arm of chromosome 17. About 50% of the cases have no family history and is thought to be caused by a new mutation. Patients with NF1 have generalized meso- and ectodermal dysplasia and demonstrate multiple subcutaneous nodules (neurofibromatosis), skin pigmentation (café-au-lait spots), neurofibromas and schwannomas of peripheral nerves, gliomas, hamartomas and neurofibrosarcomas of the central nervous system. Various skeletal abnormalities are also common and include deformity and pseudoarthrosis of the long bone, spinal scoliosis, and cystic or erosive bone changes.

Stenotic or occlusive vascular lesions are most commonly seen in renal arteries; but the aortic, celiac, mesenteric and cerebral arteries can be involved [9,10]. Spontaneous rupture and aneurysms of these arteries are also reported. The aneurysms also occur on the intracranial and the cervical cerebral arteries [10].

Surgical treatments of vertebral AVF include ligation, trapping, direct exposure and closure. Endovascular treatment using detachable balloons has been established as a standard treatment for vertebral AVF [11,12]. Endovascular treatment is easy in traumatic vertebral AVFs occurring in normal arteries, but it is difficult in high flow vertebral AVFs in NF1 because of vascular fragility, ectasia and tortuosity of the VA. In this paper, we discuss diagnostic problems, strategies and material selection for endovascular treatment of high flow vertebral AVFs in patients with NF1.

Material and Methods

A summary of the patients is presented in table 1. There were five female patients at our department from 1989 to 1997. The average age was 47.6 years (range, 29-66 years). In two initial cases (Cases 1 and 2), complete occlusion of the fistula was attained by transvenous approaches which were performed after the failure of transarterial embolisation. In the three recent cases, a staged protocol of transarterial embolisation was adopted to completely cure the fistula in the initial treatment.

Clinical Presentation

Patients most commonly presented with pulsatile tinnitus (n = 5), neck pain (n = 3), radiculopathy (n = 1), and radiculomyelopathy (n = 1).

Physical examination revealed cervical bruit (n = 5), subcutaneous neurofibroma (n = 5), café-au-lait spots (n = 4), occipital bony defect (n = 1), scoliosis (n = 1) and cervical kyphoscoliosis (n = 1).

Angiographic Findings

Six vertebral AVFs were found in five patients with NF1: three of the right and three of the left VA. The level of the fistulas were: C1 in two, C3 in one, C4 in one and C5 in two. The vertebral AVFs were supplied by the VA itself in all cases. Other feeding pedicles included the occipital artery in four, deep cervical artery in two, thyrocervical trunk (including ascending cervical artery) in three. The vertebrobasilar steal phenomenon was found in three cases. Vertebral AVFs were associated with aneurysm in the VA in four, and gigantic varices in the paravertebral venous plexus and the exits from the venous plexus in two of these patients.

Treatment Methods

Case 1, admitted to our department in 1989, was the first case we have experienced. Because of the enormous ectasia of the draining veins, it was not possible to deposit detachable balloons securely on the venous side of the fistula. Two fistulas were found on the right VA which did not show much dilatation or tortuosity (figures 1A, 1B). Therefore, three detachable balloons were placed in the right VA in an attempt to trap the fistulas; the first balloon was placed in between the fistulas, the second balloon was placed below the proximal fistula from the right VA, and the third balloon was placed above the distal fistula via the vertebral union from the contralateral VA. However, the fistulas were not completely obliterated because of the rapid development of collaterals to the right VA from the external, ascending cervical, deep cervical and the contralateral VAs.

Therefore blood flow through collaterals was reduced using detachable balloons, occlusion mini-coils and polyvinyl alcohol (PVA) particles (Contour: Boston Scientific. Co., Natick, MA). Then a huge varix, located just distal to the fistula, was catheterized by a 5F catheter using a transfemoral venous approach and embolised with 0.035 inch fibered coils (Gianturco coils: Cook Inc, Bloomington, IN) (figures 1C, 1D).

Case 2 had originally presented with a large subcutaneous haematoma in the nuchal portion because of the spontaneous rupture of a gigantic aneurysm in the left distal VA. The aneurysm was completely thrombosed following proximal occlusion of the VA with a detachable balloon. Ovoid bone defects of the left parietal bone led us to suspect NF1. However, the diagnosis was not established because of the lack of stigmas. The patient was asymptomatic for 11 years, then started complaining of pulsatile tinnitus on the left (reported elsewhere 13). Angiography revealed a relatively small fistula in the same place where there had been a gigantic aneurysm. The vertebral AVF was fed by reconstituted distal VA by collaterals between muscular branches of the proximal and distal VAs and by retrograde flow from the contralateral VA. A microcatheter was placed into a varicose vein, located just distal to the fistula, in the paravertebral plexus by transfemoral approach, and IDCs (Interlocking Detachable Coil: Boston Scientific Co., Natick, MA) were deposited to obliterate the fistula. Coil embolisation was terminated when transarterial angiography documented the disappearance of the AVF.

Biopsy showed the scalp tumours, found incidentally on CT, to be neurofibromas, and established the diagnosis of NF1 [13]. For the last three cases, a staged protocol, performed over a week, was adopted in order to attain a complete closure of the fistula in the initial treatment by the transarterial approach.

1) A microcatheter is placed into the distal stump of the VA above the fistula and the retrograde flow to the fistula is obliterated by depositing IDCs.

2) Then the tip of a microcatheter is placed into the initial part of the draining vein. The venous side of the fistula and the proximal stump of the VA are embolised until marked reduction of flow through the fistula is attained.

3) After confirming the slowing of blood flow throngh the fistula, N-butyl cyanoacrylate (NBCA) is injected to completely occlude the fistula. Relatively diluted NBCA (about 40%) is used under systemic hypotension (60 mmHg mean arterial pressure) in order to obtain sufficient penetration of the fistula.

In the last three cases, there was an extreme tortuosity and elongation of the VA. The most difficult part of the procedure was selective catheterization of the distal stump of the VA using a microcatheter. This procedure was accomplished by advancing a hydrophilic polymer-coated guide wire with a superelastic alloy deeply into the distal VA (0.016 inch Radiofocus GT guide wire, Terumo CO, Tokyo, Japan) [14]. This guide wire behaved like an anchor during the advancement of a microcatheter through an extremely tortuous VA. Another problem was a tendency of the guiding catheter to be flipped back into the subclavian artery. When this happened, it was switched to a 6/8-French coaxial guiding catheter system (Medikit co., Tokyo, Japan) which has a stronger support for a microcatheter to be advanced.

All procedures were performed using high resolution fluoroscopy and a digital subtraction angiography equipped with a road-mapping function.

Follow-up Evaluation

Follow-up angiographic examination was performed one week, three and 12 months after embolisation. Clinical follow-up was performed 2.5 to 11 years after the procedure.

Results

Embolisation Results

The vertebral AVFs were completely occluded in all cases with sacrifice of the parent artery, and the contralateral VA provided adequate blood flow to the posterior circulation. Angiographic disappearance of the vertebral AVF was confirmed immediately after embolisation in three cases and a few weeks after embolisation in one (Case 1). In one case (Case 2) occlusion was subtotal immediately after the transvenous embolisation, and it took one year for the residual fistula to be completely thrombosed.

Clinical Results

Occlusion of the afferent VAs and fistulas was tolerated without ischemic signs and symptoms in all patients. Marked improvement of neurological manifestations took place in two cases within six months of embolisation, i.e., the radiculopathy of Case 3 and radiculomyelopathy of Case 5. During follow-up evaluation for 2.5-11 years, no recurrence of symptoms or fistulas was noted in either case.

Complications

There were no transient or permanent neurological complications in any of the patients. There was a formation of a large subcutaneous haematoma in the nuchal portion of case 1 following balloon embolisation of the fistulas. This was similar to the one seen in case 2 caused by the rupture of a vertebral aneurysm. The haematoma subsided spontaneously within a week.

Illustrative Cases

Case 1

A 41-year-old female with NF1 complained of pulsatile tinnitus on the right for two years. Physical examination demonstrated numerous cutaneous nodules and café-au-lait spots all over the trunk. The neurological examination was unremarkable.

Angiography showed high-flow vertebral AVFs in the right distal VA, however, angioarchitecture was not obvious due to the rapid transit of contrast material into the huge varices through the fistula. Angiography under flow control by use of a balloon catheter revealed two fistulas at the levels of C1 and C3. Steal phenomenon from the left VA to the distal fistula was prominent (figures 1A, 1B). Trapping of the two fistulas was performed using three detachable balloons. This procedure resulted in temporary relief of the tinnitus and formation of a large subcutaneous haematoma in the nuchal portion.

However, the tinnitus recurred within one week after the procedure when the subcutaneous haematoma subsided. Follow-up angiography revealed a recanalization of the fistulas via the external carotid and vertebral branches.

The external carotid, ascending and deep cervical arteries, which were giving off collateral vessels to the muscular branches of the VA, were embolised using microcoils, small detachable balloons and PVA particles. However, the fistulas were kept opacified mainly via the collateral vessels from the opposite occipital artery (figure 1C). A huge varicose vein located just distal to the fistula was successfully cannulated by a 5 French catheter advanced through the deep cervical vein by transfemoral approach. The varix was embolised with multiple fibered Gianturco coils (0.035 inch) and the fistula was completely occluded (figure 1D).

The angiographic follow-up, performed after six months, one, two and three years, did not show any recanalization of the fistula. The patient has been doing well for the last 11 years.

Case 5

A 51-year-old female with NF1 presented with hypesthesia of the left arm in 1989 and then of the right in 1993. The patient developed gait disturbance in 1994 because of weakness of the lower extremities. When she was admitted to a local hospital for the operation of myoma uteri, she was diagnosed as having a vertebral AVF on cervical MRI. On T2 weighted images (figures 2A, 2B), MRI showed a prominent cervical kyphosis due to a compression fracture of the C5 and C6 vertebral body and large ovoid flow voids in the para- and prevertebral region. However, no treatment was performed. There was gradual progression of paraparesis, muscle atrophy and hypesthesia of both arms by the end of 1995. She had marked difficulty in walking and urinary incontinence by the spring of 1996. She was admitted for endovascular treatment to an outside hospital in October 1996.

Angiography showed the right VA to be markedly dilated and extremely tortuous with a fistula draining into gigantic varices at C4 level. There was a subsequent filling of the paravertebral venous plexus and right internal jugular vein carrying several varices. The right VA was not opacified above the fistula. Additional blood flow came to the fistula through the right ascending cervical, deep cervical and occipital arteries and segmental branches of the left VA. These arteries anastomosed with the distal right VA and provided additional flow to the fistula in retrograde fashion (figures 2C, 2D). Endovascular treatment performed at

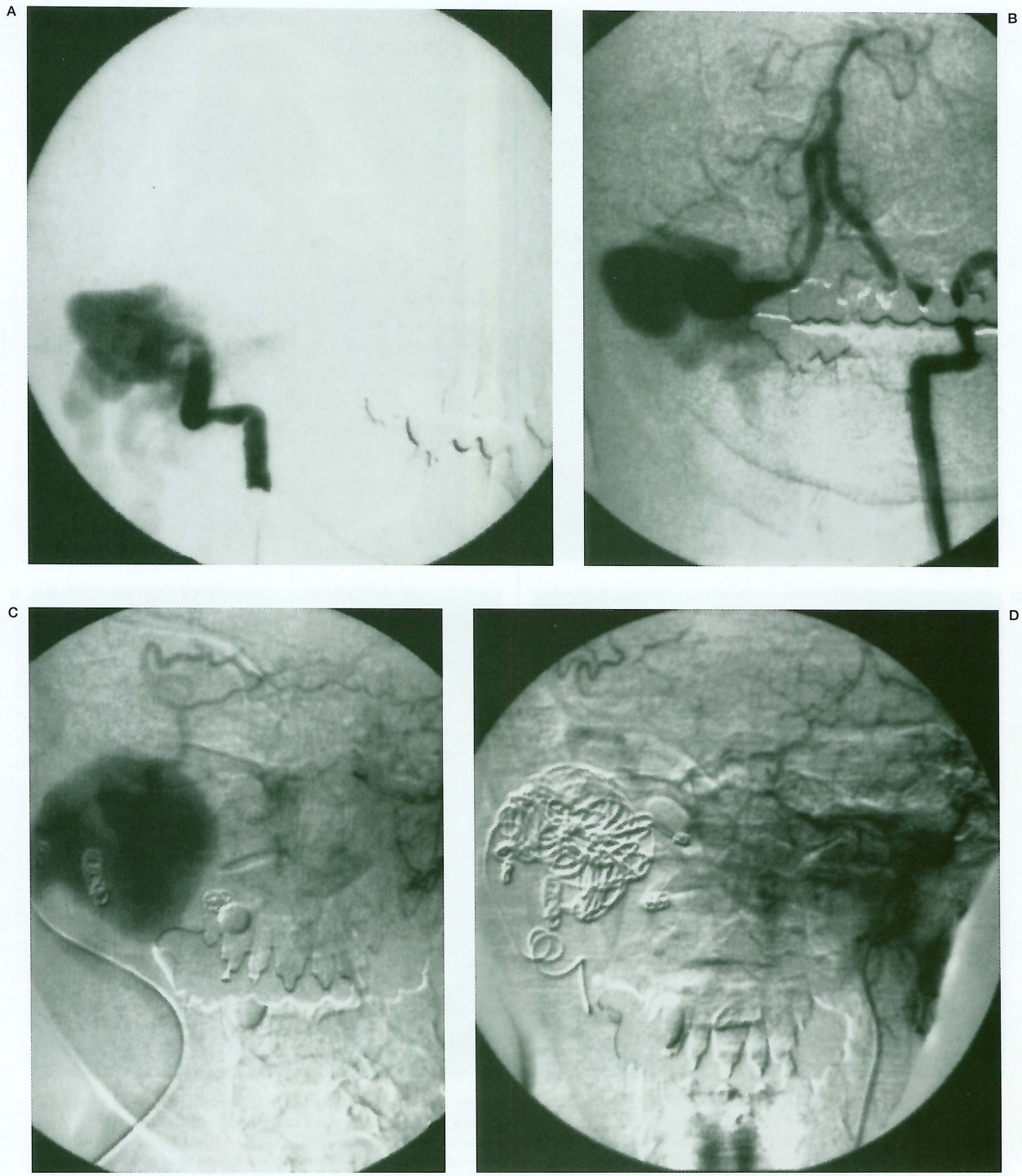

Figure 1 Case 1. Extremely high flow dual vertebral AVFs, associated with gigantic varices, on the right. A) Right VAG, AP projection, demonstrating a proximal fistula at the C3 level. B) Left VAG, AP projection, showing retrograde filling of the distal fistula at the C1 level. Left external carotid injection, AP view, before (C) and after (D) transvenous embolisation. Following failed trapping procedure of the vertebral AVF using detachable balloons (black arrows), the right external carotid branches are extensively embolised using coils (white arrows) and PVA particles. Then transvenous embolisation is performed. C) The left occipital artery injection, venous phase, showing a large varix opacified through collaterals. Note a 5 French catheter placed in the varix via the transfemoral approach. D) The same injection following transvernous embolisation using fibered 0.035 inch occlusion coils. Note that the varix is no longer opacified as the medial part of the varix, connected to the fistula, is densely packed.

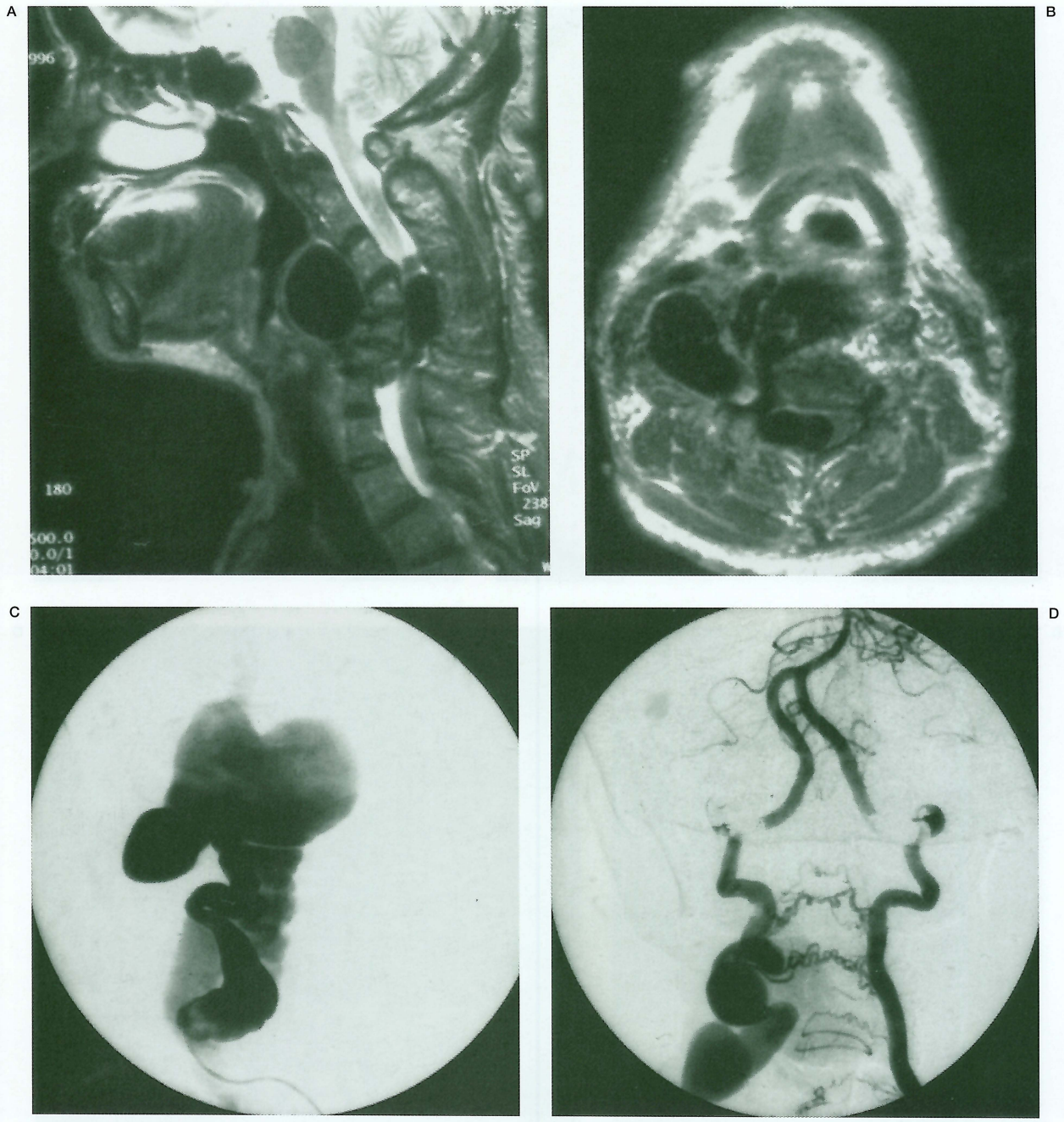

Figure 2 Case 5. A) Sagittal T2 weighted MRI demonstrating severe cervical kyphoscoliosis and intraspinal and prevertebral ovoid masses with flow void. B) Axial T1 weighted MRI showing severe spinal cord compression by a varix (white arrows) which is contiguous with prevertebral varices through the neural foramen. C) Right VAG, AP projection, showing a markedly tortuous and dilated VA, a fistula at C4 level and gigantic varices. D) Left VA injection showing a prominent steal phenomenon to the high flow right vertebral AVF. A result of a failed attempt to trap the vertebral AVF performed at an outside hospital.

an outside hospital ended up in incomplete trapping of the fistula after losing several detachable balloons into the venous circulation (figures 2E, 2F). She was referred to our hospital for the complete endovascular treatment on January 22, 1997. Physical examination showed numerous cutaneous neurofibromas and café-au-lait spots all over the body. Neurological examination showed radiculomyelopathy consisting of hypesthesia below C6 level, muscle atro-

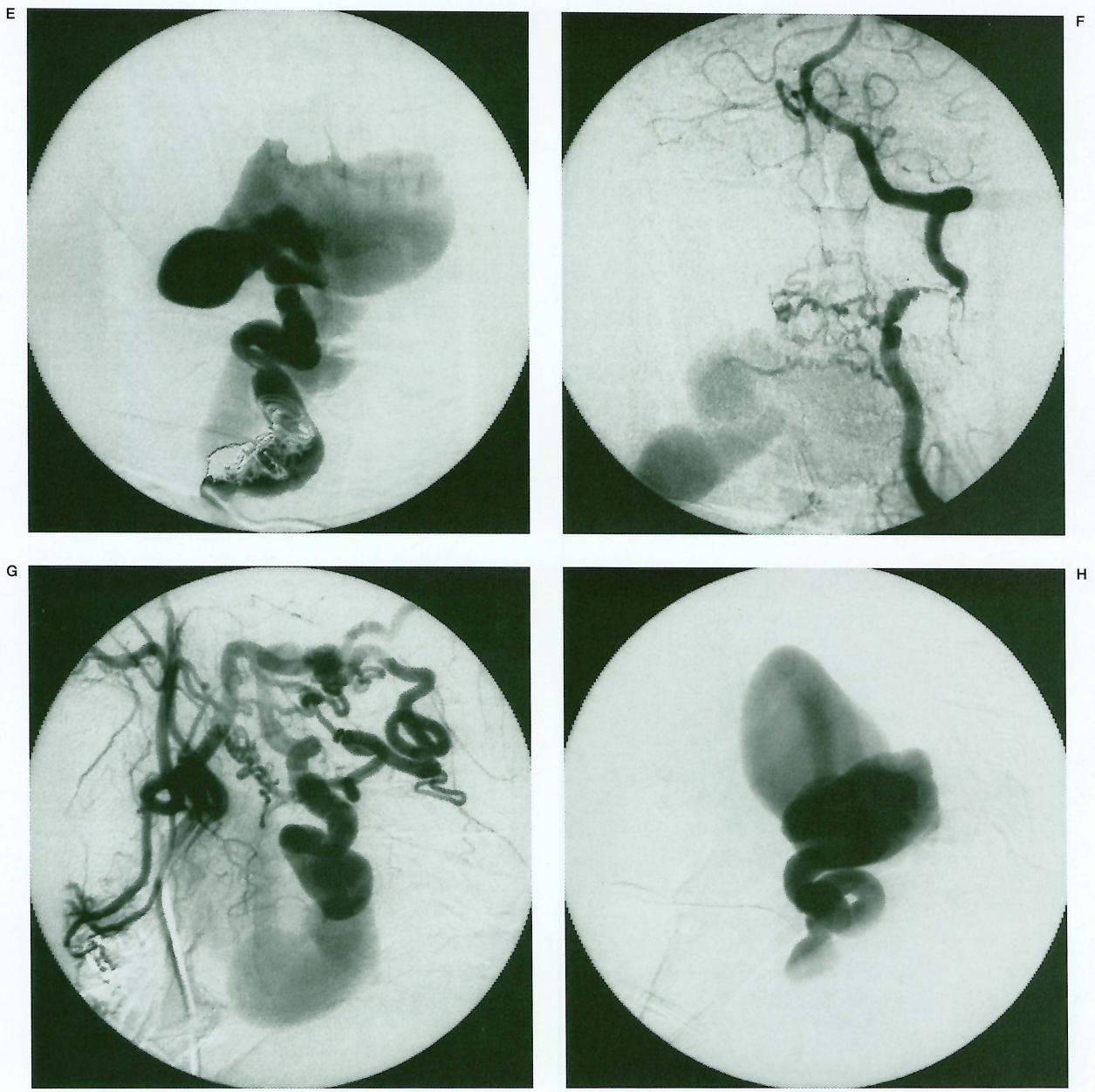

Figure 2 Case 5. E) Right VAG, AP view, showing a mass of occlusion coils placed in the initial part of the right VA. F) Left VA injection showing a balloon detached in the most distal part of the right VA, and opacification of the fistula via radicular arteries of the left VA. This balloon was inadvertently detached while being advanced into the right VA in retrograde fashion from the contralateral VA via the vertebro-vertebral junction at the outside hospital. G) Right external carotid angiogram, lateral projection, showing a tortuous and dilated distal VA and a fistula opacified in retrograde fashion via the collaterals arising from the occipital artery. H) Right VAG, lateral projection, showing a serpentine VA, the fistula, the first and the second varices.

phy of the upper extremities, a generalized hyperreflexia and pathological reflexes in four extremities.

Great difficulty was encountered during selective cannulation into the distal stump of the right VA because of a tremendous frictional resistance generated between the inner wall of the VA and the surface of the microcatheter due to a prominent tortuosity of the artery (figures 2G, 2H). By adopting a triple axial

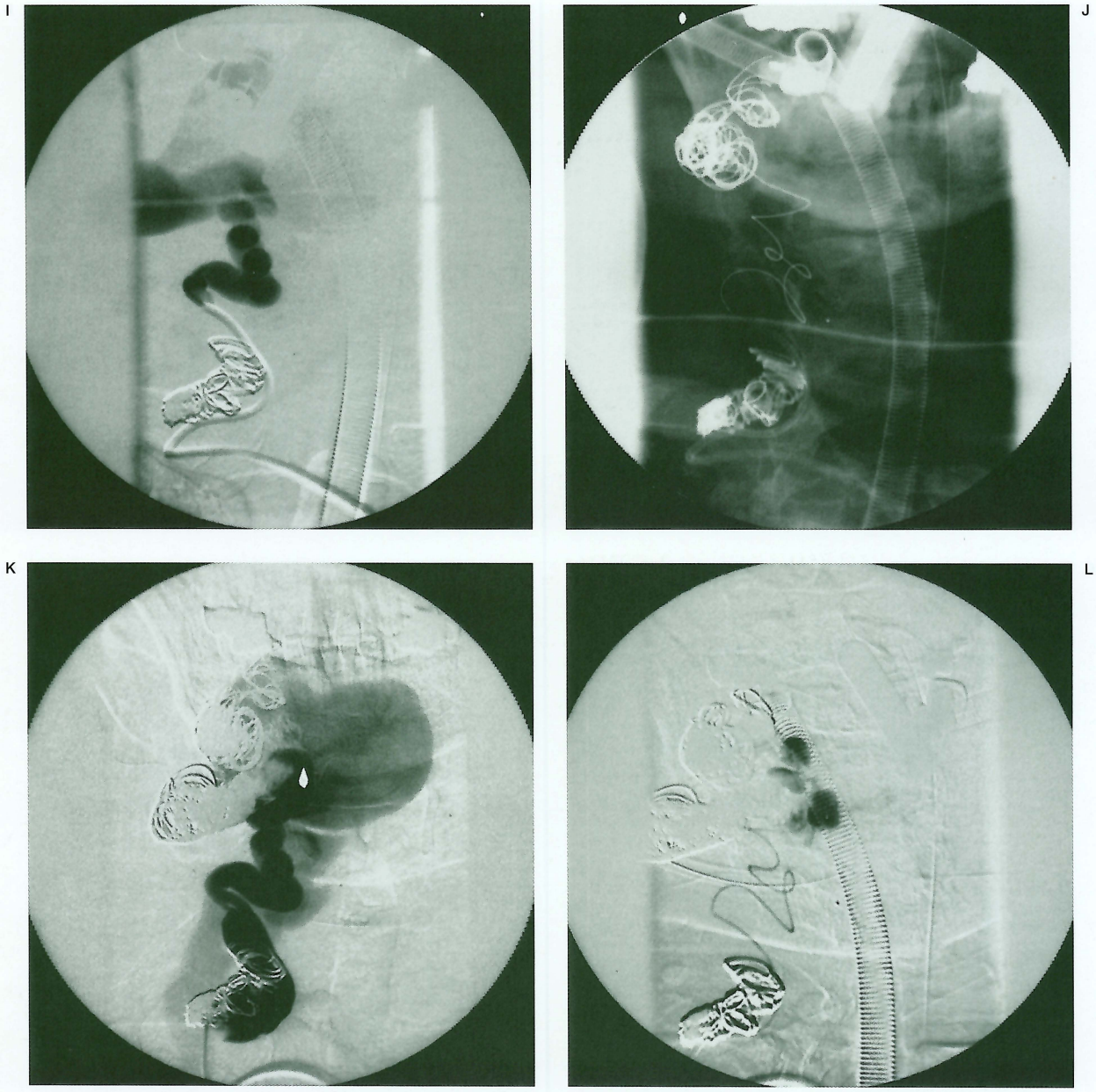

Figure 2

catheter system consisting of a microcatheter and a 6/8 French coaxial guiding catheter, superselective cannulation into the distal VA was attained with strong support from the introducing catheter. Then, the distal stump of the right VA was obliterated with IDCs (figures 2I, 2J). The second session of embolisation was performed a week later. A microcatheter was advanced from the proximal stump of the VA to the varix formed at the initial part of the draining vein through the fistula. These vascular components were roughly packed with IDCs (figure 2K). After confirming the slowing of blood flow through the fistula, NBCA was injected under systemic hypotension to occlude the fistula (figure 2L).

The radiculomyelopathy showed a marked recovery after the procedure and the patient

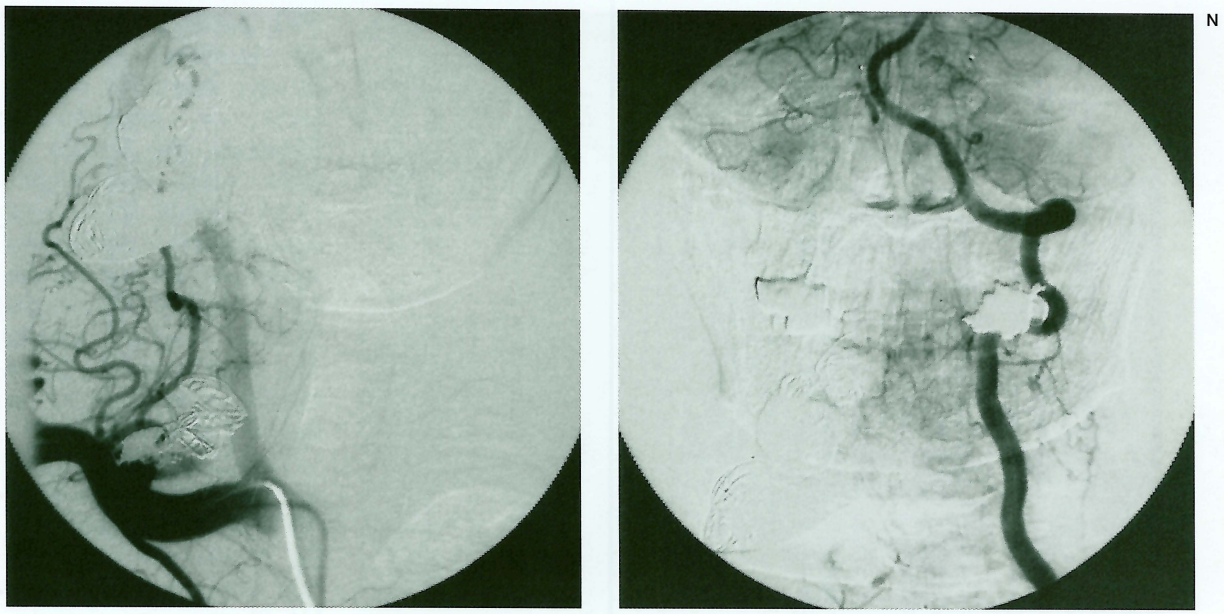

Figure 2 Case 5. I) Right VAG, AP projection, showing a 6/8 French coaxial guiding catheter advanced beyond the coils placed in the proximal part of the VA. A microcatheter can be placed into the distal stump of the VA due to strong support from the guiding catheter. J) Coils being placed into the aneurysm on the distal stump of the VA. Note an extreme tortuosity of the VA. K) Right VAG, AP view, showing denser opacification of the VA just proximal to the fistula generated by marked decrease of flow through the fistula after the placement of coils in the varix and the proximal stump of the VA. L) A column of NBCA injected through a microcatheter, which was placed in the proximal stump, stops within a few centimeters in the draining vein after penetrating the fistula. Control angiography, M) right subclavian injection, AP view obtained a week after embolisation, showing complete obliteration of the fistula. N) Left VAG, AP view. No fistula is opacified either.

was walking normally at one year follow-up. Control angiography showed obliteration of the fistula persisted (figures 2M, 2N).

Discussion

Diagnosis of NF1 was easy in four of our cases because of the existence of multiple subcutaneous nodules (neurofibromatosis) and skin pigmentation [15]. Various skeletal abnormalities were seen in three of our cases.

In case 2, these stigmas were all lacking. The only clue to the diagnosis was the skull abnormality. Although skull abnormalities are uncommon compared to the involvement of the axial and peripheral skeleton, they may occasionally include bizarre deformities of the calvarium and facial bones, erosion and enlargement of skull foramina (especially due to cranial nerve neurofibromas) and ovoid bone defects of the skull vault. The greater and lesser sphenoid wings are most commonly involved, resulting in changes of the orbital walls and sella turcica, sometimes with associated exophthalmos [16]. As was seen in our cases 3-5 (figures 2A, 2B), the most characteristic MRI findings of vertebral AVFs in NF1 were cervical spine kyphosis with spinal cord angulation and prevertebral oval masses with signal loss consistent with vascular structures [17].

Thirty cases with vertebral AVFs with NF1 were identified in the literature including our five cases [8,18-22]. The mean age was 42.1 years (range: 11 to 66 years). There were 24 females and 6 males. The left VA was involved in 16 patients, the right in 12, and two patients developed bilateral AVFs. Pulsatile tinnitus, neck pain and cervical bruit were the common clinical manifestations. Neurological signs or symptoms were present in 80% of the patients. Direct compression of the nerve roots and spinal cord could be the primary mechanism for neurological symptoms as was seen in our Cases 4 and 5. AVFs involved all segments of the cervical VA. In high flow vertebral AVFs, abundant flow was recruited to the fistula from the ascending cervical, deep cervical and occipital arteries which reconstituted distal VAs.

Cluzel et Al described spontaneous vertebral AVFs comparing 19 NF1 patients with 31 non-

Table 1 **Summary of our cases**

Case	Age Sex	Side level	Clinical presentation	Physical findings	Endovascular treatment approach	material	results	complications	Last follow-up angiographical	clinical
1	41 F	Rt C1 & 3	Pulsatile tinnitus	bruit subcutaneous nodules café-au-lait spots	TA + TV	DB mini coil Gianturco coil	Complete occlusion	Large nuchal haematoma	Cure (3 years)	Intact (11 years)
2*	51 F	Lt C1	Pulsatile tinnitus	bruit scalp soft tissue mass occipital bony defect	TA + TV	PVA IDC	Complete occlusion	None	Cure (2.5 years)	Intact (4.8 years)
3	29 F	Lt C5	Pulsatile tinnitus neck pain	bruit subcutaneous nodules café-au-lait spots scoliosis	TA	IDC NBCA	Complete occlusion	None	Cure (1 year)	Intact (3.5 years)
4	66 F	Lt C5	Pulsatile tinnitus neck pain radiculopathy	bruit subcutaneous nodules café-au-lait spots	TA	IDC NBCA	Complete occlusion	None	Cure (1 year)	Intact (2.5 years)
5	51 F	Rt C4	Pulsatile tinnitus neck pain radiculomyelopathy	bruit subcutaneous nodules café-au-lait spots kyphoscoliosis	TA	IDC NBCA	Complete occlusion	None	Cure (1 year)	Intact (3.2 years)

Rt = right; Lt = left; TA = transarterial; TV = transvenous; DB = detachable balloon; PVA = polyvinyl alcohol; IDC = interlocking detachable coil; NBCA = N-Butyl Cyanoacrylate – Presented elsewhere * = Surg Neurol 51: 168-173, 1999

NF1 patients. There was no significant difference in age, laterality or the level of involvement. However, the female to male ratio was higher (5.3:1 versus 2.1:1) and neurological symptoms were more common in the NF1 group (73.6% versus 22.5%). Venous ectasia was much more frequent (52.6% versus 14%) in the NF1 group[8].

The ideal goal of treatment of the vertebral AVF is the occlusion of the fistula and preservation of the patency of the VA. Treatment options include surgical ligation, trapping, direct exposure and closure, or transcatheter embolisation (arterial and/or venous approach). Surgical treatment of vertebral AVFs, however, has often been unsatisfactory and is always difficult, because of the extensive anastomotic network between the external carotid, subclavian and contralateral VAs and the affected VA [8,23]. Such collaterals most frequently arise from the ipsilateral ascending cervical artery of the thyrocervical trunk, since embryologically this vessel and the VA both originate as longitudinal anastomoses between the first six pairs of embryonic dorsal intersegmental arteries, which are paired branches of the primitive aortic arches[24].

Endovascular treatment of vertebral AVFs by detachable balloon has been praised as a minimally invasive and the most reli-

able method for occluding the fistula [1]. However, preservation of the parent artery is easy only in traumatic vertebral AVFs occurring in the normal artery. It is very difficult to obliterate vertebral AVFs associating NF1 using detachable balloons, because of increased fragility, ectasia and tortuosity of the VA and gigantic varices in draining veins [5]. Furthermore, Simionate et Al reported a case with recurrence of a massive intracranial haemorrhage caused by embolisation of vertebral AVF by placing four detachable balloons in the fistula in an attempt to preserve the VA. They considered that the cause of the haemorrhage was an exacerbation of ascending venous flow from the fistula by the inappropriate position of the balloons, and recommended endovascular direct occlusion of the VA to treat high flow vertebral AVF (F. Simionato, poster presentation at the World Federation of Interventional and Therapeutic Neuroradiology, Vilamoura, Portugal, 1999).

Trapping procedures of the involved segment of the VA, by placing detachable balloons on both distal and the proximal sides of the fistula, are not justified because of the existence of numerous potential feeders which will quickly recanalize the fistula. In addition, once direct transarterial access to the fistula has been obliterated by unsuccessful occlusion, there is no arterial route available to attain cure by embolisation [24].

When the fistula is not completely obliterated by the transarterial approach, transvenous occlusion of the fistula is the alternative treatment if it is possible to reach the fistula by transvenous approach, as was shown in our two initial cases [25].

The most suitable embolic material currently available to attain complete closure of the AVFs occurring on fragile arteries by transarterial approach is a liquid adhesive, namely NBCA. However, it is difficult to deposit NBCA accurately in the high flow fistula, because there is great danger of NBCA passing through the fistula and causing pulmonary embolism [9].

Therefore, a preliminary procedure to decrease flow by placing coils including Guglielmi Detachable Coils, IDC and fibered coils is necessary.

Although detachable coils are composed of bare platinum coils and may be less thrombogenic, accurate and compact packing of the blood vessel can be performed safely through microcatheters. By combining detachable coil placement with NBCA, total obliteration of the fistulas becomes possible in a staged fashion as follows:

1) embolisation of the distal stump of the VA with detachable coils;

2) embolisation of the initial part of the varicose vein and the proximal stump of the VA with detachable coils. (Ideal flow for depositing NBCA can be created by the amount of coils deposited), and

3) a complete closure of the fistula by injecting NBCA under systemic hypotension (60 mmHg mean arterial pressure).

In conclusion, complete obliteration of vertebral AVFs in cases with NF1 can be attained by adopting a staged protocol, as was performed in Cases 3, 4 and 5 without any transient or permanent complications.

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